Virulence potential and adherence properties of *Escherichia coli* that produce CTX-M and NDM $\beta$-lactamases

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The success of certain sequence types such as ST131 that produce CTX-M or NDM $\beta$-lactamases, and ST405 that produce CTX-M $\beta$-lactamases, among extraintestinal *Escherichia coli* (ExPEC) had previously been linked to a combination of antimicrobial resistance and certain virulence factors. The adherence properties of these sequence types to gastro-intestinal epithelial cells had not been investigated. A study was therefore designed to investigate the phylogenetic groups, virulence factors and adherence properties of *E. coli* sequence types ST101, ST131 and ST405 that produce CTX-M-15 and NDM-1. Our results show that ST131 was positive for phylogenetic group B2, ST101 for B1 and ST404 for D. ST131 had more virulence factors than ST101 or ST405. Interestingly, ST101 adhered more avidly to HEp-2 and Caco-2 cells than did ST131 and ST405. Our study showed that adherence to gastro-intestinal cells did not seem to play an important role in the worldwide epidemiological success of ST131 and ST405. The exact role of ExPEC-associated virulence genes is unknown and it is unlikely that one set of factors determines the virulence properties and epidemiological success of certain sequence types. Future investigations should be undertaken to study the microbiological and ecological factors that make certain sequence types among ExPEC such successful pathogens.

INTRODUCTION

*Escherichia coli* is an important cause of community and hospital-associated human infections and is responsible for considerable morbidity, mortality and increased health costs (Russo & Johnson, 2003). The management of infections due to extraintestinal *E. coli* (ExPEC) has been complicated by the emergence of antimicrobial resistance, especially since the late 1990s (Pitout & Laupland, 2008). $\beta$-Lactamases are bacterial enzymes that inactivate $\beta$-lactam antibiotics by hydrolysis, which results in ineffective compounds. Most important within ExPEC is the increasing recognition of isolates producing ‘newer’ $\beta$-lactamases that consist of extended-spectrum $\beta$-lactamases (ESBLs) (e.g. CTX-M types) and carbapenemases [e.g. metallo-$\beta$-lactamases (MBLs) such as the NDMs, serine $\beta$-lactamases such as KPCs and OXA-48-like enzymes] (Pitout, 2012). Since the late 1990s, CTX-M types of ESBL enzymes have emerged worldwide among *E. coli*, and have become the most widespread type of ESBL in the world (Pitout & Laupland, 2008). A new type of MBL, named NDM, has been described in *Klebsiella pneumoniae* and *E. coli* recovered from a Swedish patient who was previously hospitalized in New Delhi, India (Yong et al., 2009). Recent reports from the Indian subcontinent (including India, Pakistan and Bangladesh) show that the distribution of NDM $\beta$-lactamases among *Enterobacteriaceae* is widespread in these countries, while sporadic cases of infections due to bacteria with these enzymes have been reported from different parts of the world, including several countries in Europe, North America, the Middle East, Asia, Africa and Australia (Nordmann et al., 2011).

Multilocus sequence typing (MLST), which uses sequence variation in a number of housekeeping genes to define sequence types or clones, is an excellent tool for evolutionary studies to show common ancestry lineages among bacteria (Sullivan et al., 2005). It has led to the definition of major sequence types (STs) and the recognition of successful widespread international antimicrobial-resistant sequence types such as *E. coli* ST131 and ST405 that produce CTX-M $\beta$-lactamases (Peirano & Pitout, 2010). ST131 is prevalent in most parts of the world, and is often associated with fluoroquinolone resistance and the production of CTX-M-15.

Abbreviations: ESBL, extended-spectrum $\beta$-lactamase; ExPEC, extra-intestinal *Escherichia coli*; MBL, metallo-$\beta$-lactamase; MLST, multilocus sequence typing; ST, sequence type.
### Table 1. Susceptibilities and molecular characteristics of CTX-M-15- and NDM-1-producing E. coli

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</table>
ST405 with various types of CTX-M β-lactamases also has a worldwide distribution but is not as prevalent as ST131 (Coque et al., 2008; Jones et al., 2008; Mihaila et al., 2010; Smet et al., 2010). These reports suggest that the intercontinental dissemination of ST131, and to a lesser extent ST405, has in part contributed to the worldwide emergence of CTX-M-producing E. coli. blaNDM genes have also been identified in internationally successful sequence types such as E. coli ST101 (Mushtaq et al., 2011) and ST131 (Peirano et al., 2011b).

The success of ST131 and ST405 among ExPEC has been linked to a combination of antimicrobial resistance and certain virulence factors (Johnson et al., 2010; Van der Bij et al., 2012); however, to our knowledge the adherence properties of these sequence types to gastro-intestinal epithelial cells have not been investigated. A study was therefore designed to investigate the phylogenetic groups, virulence factors and adherence properties of E. coli sequence types ST101, ST131 and ST405 that produce CTX-M-15 and NDM-1.

### METHODS

#### Bacterial isolates.

The following E. coli isolates were determined in this study: MH01 (ST101 with NDM-1), EC271 (ST101 with NDM-1), CH01 (ST131 with NDM-1), EC57 (ST131 with CTX-M-15, OXA-1 and TEM-1) and EC27 (ST405 with CTX-M-15). MH01 and CH01 were isolated from the urine of patients with lower urinary tract infections (Peirano et al., 2011a, b; Poirel et al., 2010) while EC57 and EC27 were isolated from blood of patients with upper urinary tract infections (Peirano et al., 2012).

#### Antimicrobial susceptibility testing.

MICs were determined with the Microscan NEG MIC 30 panel (Siemens) and custom-made microdilution panels (PML Microbiologicals). The following antimicrobial agents were tested: amoxicillin-clavulanic acid (AMC), piperacillin-tazobactam (TZP), cefoxitin (FOX), ceftriaxone (CRO), ceftazidime (CAZ), aztreonam (AZT), imipenem (IPM), meropenem (MEM), amikacin (AMK), gentamicin (GEN), tobramycin (TOB), ciprofloxacin (CIP) and trimethoprim-sulfamethoxazole (SXT). Throughout this study, results were interpreted using CLSI (2012) criteria for broth dilution. The quality-control strains used for this part of the study were E. coli ATCC 25922, E. coli ATCC 35218 and Pseudomonas aeruginosa ATCC 27853.

#### β-Lactamase identification.

PCR amplification sequencing for blaCTX-M, blaOXA-24, blaTEM and blaNDM was carried out on the isolates with a GeneAmp 9700 Thermocycler instrument (Applied Biosystems) using PCR conditions and primers as previously described (Peirano et al., 2011a, b; Pitout et al., 2007).

#### Plasmid-mediated quinolone-resistance determinants.

The amplification of the qnrA, qnrS and qnrB genes was undertaken in all the isolates by multiplex PCR (Robicsek et al., 2006). aac(6’)-Ib and qepA were amplified in a separate PCR using primers and conditions as previously described (Park et al., 2006; Yamane et al., 2008). The variant aac(6’)-Ib-cr was further identified by digestion with BsaFI (Yamane et al., 2008) (New England Biolabs).

#### Identification of sequence types.

MLST was performed using seven conserved housekeeping genes of E. coli (adk, fumC, gyrB, icd, mdh, purA and recA). A detailed protocol of the MLST procedure, including allelic type and sequence type assignment methods, is available at MLST Databases at the ERI, University College Cork website (http://mlst.ucc.ie/mlst/dbs/Escoli).

#### Virulence factors.

The presence of ExPEC-associated virulence genes was assessed by multiplex PCR (Johnson et al., 2009). Isolates were defined as ExPEC if positive for two or more of papA and/or papC (P fimbiae major subunit and assembly), sfa/focDE (S and F1C fimbiae), afa/draBC (Dr-binding adhesins), kpsM II (group 2 capsule) and iutA (aerobactin receptor) (Johnson & Stell, 2000). The virulence score was the number of virulence genes detected, adjusted for multiple detection of the pap, sfa, foc and kpsM II operons.

#### HEp-2 and Caco-2 cell early localized adherence assay.

HEp-2 and Caco-2 cells were cultured in MEM + 10% fetal bovine serum on 12 mm coverslips in 24-well tissue culture plates at 1 × 10⁵ and 2.5 × 10⁵ cells ml⁻¹ (1 ml per well), respectively, at 37 °C, 5% CO₂ overnight. Overnight bacterial cultures were normalized to OD₆₀₀ and subcultured 1:100 into DMEM for 60 min at 37 °C, 5% CO₂. Normalized bacterial cultures were then added to the HEp-2 or Caco-2 cells for 30 min at 37 °C, 5% CO₂ in the presence of 0.5 M mannose to inhibit type 1 fimbiae-mediated adherence, as described in our previous articles (Hyland et al., 2006, 2008). The cells were

### Table 1. cont.

<table>
<thead>
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*See Methods for full names of antibiotics.

†F10 papA, P fimbiae subunit variant; papACEFG, genes of P fimbiae operon; sfa/foc, S or F1C fimbiae; focG, F1C fimbiae adhesin; iha, adhesion siderophore; fimH, type 1 fimbiae; tsh, temperature-sensitive haemagglutinin; hra, heat-resistant agglutinin; afa/dra, Dr-binding adhesins; hlyD, α-haemolysin; sat, secreted autotransporter toxin; pic, serine protease; vut, vacuolating toxin; astA, enteroaggregative E. coli toxin; cnf1, cytotoxic necrotizing factor; iroN, salmonellin (siderophore) receptor; fyuA, yersiniabactin (siderophore) receptor; treA, siderophore receptor; iutA, aerobactin (siderophore) receptor; kpsM II, group 2 capsule; K1, K2, and K5, group 2 capsule variants; kpsMT III, group 3 capsule; iop, uropathogenic-specific protein; truT, serum resistance-associated; ompT, outer-membrane protease T; iss, increased serum survival; H7 flic, flagellin variant; malX, pathogenicity island marker.

‡The virulence score is the number of virulence genes detected, adjusted for multiple detection of the pap, sfa, foc and kpsM II operons.
then washed five times with PBS and fixed with methanol. They were next stained with Giemsa, and bacterial adhesion was assessed by two observers blinded to the sample identities. Approximately 150 cells were observed for each condition, counting the number of bacteria per HEp-2 or Caco-2 cell. The assay was repeated five times to ensure reproducibility.

RESULTS AND DISCUSSION

The present study investigated the antimicrobial resistance, virulence-associated traits and adherence properties of ST131 that produce NDM-1 and CTX-M-15, ST101 that produce NDM-1, and ST405 that produce CTX-M-15. The susceptibilities, β-lactamases, plasmid-mediated quinolone-resistance determinants, sequence types, phylogenetic groups and virulence factors are shown in Table 1. CH01 and EC57 (both ST131) were positive for the following virulence factors: F10 papA (P fimbriae subunit variant), iha (adhesion siderophore); fimH (type 1 fimbriae), sat (secreted autotransporter toxin), fyuA (yersiniabactin (siderophore) receptor), iutA [aerobactin (siderophore) receptor], kpsM II (group 2 capsule) K2 (group 2 capsule variants), usp (uropathogenic-specific protein), traT (serum resistance-associated), ompT (outer-membrane protease T) and malX (pathogenicity island marker). MH01 and EC271 (both ST101) were positive for papEFG (P fimbriae subunit variants), sfa/foc (S or F1C fimbriae), fimH (type 1 fimbriae), hra (heat-resistant agglutinin), fyuA (yersiniabactin (siderophore) receptor) and usp (uropathogenic-specific protein). EC27 (ST405) was positive for fimH (type 1 fimbriae), fyuA (yersiniabactin receptor), iutA (aerobactin receptor), K2 (group 2 capsule variants), kpsMT III (kpsMT III, group 3 capsule), traT (serum resistance-associated), and malX (pathogenicity island marker) (Table 1).

Our results show that ST131 had more virulence factors than ST101 or ST405. The virulence score of ST131 was the highest, while ST101 was the lowest. ST131 and ST405 were positive for ExPEC (Table 1). Johnson et al. (2010) investigated the presence and virulence properties of ST131 among 127 ExPEC, and also showed that the combination of antimicrobial resistance and virulence factors most likely give ST131 a competitive advantage over other isolates of E. coli, promoting its clonal expansion and dominance over less virulent and/or more susceptible E. coli clones (Johnson et al., 2010).

Van der Bij et al. (2012) recently determined the virulence profile of ST405 and also showed that the combination of antimicrobial resistance, phylogenetic group D and the presence of certain virulence factors such as aerobactin receptor (iutA), serum-resistance-associated factor (traT) and pathogenicity island marker (malX) might be important in the worldwide spread of this sequence type. Our results regarding the virulence factors of ST131 and ST405 support the findings of Johnson et al. (2010) and Van der Bij et al. (2012) and it seems that certain virulence factors, such as sat, iutA, malX, usp and ompT, might increase the adaptability, competitiveness and the ability to efficiently colonize the human body of certain sequence types.

ST131 strains that produce CTX-M-15 have emerged since 2003 as important pathogens causing worldwide community-associated infections (Pitout et al., 2005). A study from Calgary demonstrated that travel to the Indian subcontinent was associated with high risk of community-onset infections with ESBL-producing E. coli in returning travellers (Laupland et al., 2008). A follow-up study from the same group showed that these infections were mostly due to the acquisition of ST131 that produce CTX-M-15.
Table 2. Quantitative localized adherence assay with HEP-2 cells of CTX-M-15- and NDM-1-producing E. coli

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</table>

(Pitout et al., 2009). The initial rectal colonization of travellers with CTX-M-producing E. coli (especially those that belong to ST131) and the subsequent worldwide distribution most likely occurred during visits to the Indian subcontinent from the late 1990s and early 2000s (van der Bij & Pitout, 2012). Since recent reports from India have indicated that more than 70% of E. coli collected from the community are ESBL producers (Hawser et al., 2009), foreign travel to high-risk areas such as the Indian subcontinent may have played an important role in rectal colonization of travellers and subsequent spread of CTX-M-producing E. coli across different continents.

CH01 (ST131) with NDM-1 (papA+, iha+, sat+, fimH+) and EC57 (ST131) with CTX-M15, OXA-1, TEM-1 (papA+, iha+, sat+, fimH+) adhered the least to HEP-2 and Caco-2 cell monolayers (Figs 1 and 2, Table 2). These results do not support the notion that adherence to gastrointestinal cells played an important role in the worldwide epidemiological success of ST131. A limitation of this study is that we only tested two isolates of ST131. We recommend that more isolates that belong to ST131 be tested for their adherence properties.

MH01 and EC271 (ST101) with NDM-1 and CTX-M-15 (papEF+, sfa/foc, fimH+, hra+) adhered the most avidly to both HEP-2 (mean of 7.86 and 10.44 respectively) and Caco-2 (mean of 8.36 and 8.52 respectively) cell monolayers (Figs 1 and 2, Table 2). MH01 and EC271 belonged to phylogenetic group B1 and were positive for 4/10 adhesin types of virulence factors (i.e. papEF, sfa/foc, fimH, hra). This could explain the avid adherence of these isolates (Table 2). E. coli ST101 was first described in Spain among ExPEC that produced CTX-M-14 (Coelho et al., 2011; Mora et al., 2011). Subsequently, ST101 with NDM-1 was detected in Australia from a patient previously admitted to a hospital in Bangladesh (Poirel et al., 2010), in Canada from a patient previously admitted to a hospital in India (Peirano et al., 2011), and from patients in Pakistan and the UK (Mushtaq et al., 2011).

Our study shows that the exact role of ExPEC-associated virulence genes and adherence factors is unknown and it seems unlikely that one set of factors determines the virulence properties and epidemiological success of certain sequence types (Dobrindt, 2005). Future investigations should be undertaken to further study the microbiological and ecological factors that make certain sequence types such as ST131 among E. coli such successful pathogens.

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REFERENCES


